computed tomography (HRCT) in "healthy" smokers and its relation to neutrophil-associated inflammatory markers in bronchoalveolar lavage (BAL) and blood.

Material and Methods: We recruited 30 "healthy" smoking and 18 never-smoking men from a population study "Men born 1933 in Göteborg". A HRCT, a bronchoscopy with a BAL and blood tests were done. HRCT was analysed visually. We analysed myeloperoxidase (MPO), interleukin-8 (IL-8) and human neutrophil lipocalin (HNL) in both BAL and blood.

Results: Emphysematous lesions were demonstrated in 13/30 smokers and in 1/18 never-smokers.

Correlations – emphysema to inflammatory markers in BAL

	MPO	IL-8	HNL
r	0.52	0.57	0.76
p	n.s.	0.04	0.006

Spearman Rank correlation test

No correlations were seen between emphysema and inflammatory markers in blood.

Conclusion: In a sample of "healthy" smokers there are emphysematous lesions that are correlated to mainly HNL, a neutrophil activation marker in BAL, indicating that neutrophils has a role in the pathogenesis of emphysema.

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P2041
EFFECT OF INHALED STEROIDS ON CELLS AND MOLECULAR MEDIATORS OF AIRWAY INFLAMMATION IN COPD

B. Balbi[1], M. Majori[2], S. Bertacco[2], G. Convertino[1], A. Cuomo[2], C.F. Donner, A. Pesci[2]. *S. Maugeri Foundation, IRCCS, Rehabilitation Institute of Veruno (NO); [1]Section of Varallo S. (VC); [2]Respiratory Diseases Dep., University of Parma, Italy*

Although treatment with inhaled steroids is widely used in COPD, few studies have investigated its effects on airway inflammation (AI). To look for changes in the cellular and molecular mediators of AI, we performed bronchoscopy and bronchial lavage (BL) in 8 current smokers with stable COPD 0 (FEV_1 69.8 ± 2.1% predicted) before and after a 6-week treatment with inhaled beclomethasone (1.5 mg/day). Ten normal persons served as controls. In BL total and differential cell counts and determination of the levels of interleukin-8 (IL-8), myeloperoxidase (MPO), eosinophilic cationic protein (ECP) and tryptase were done. In addition the Symptom Score (SS), the endoscopic Bronchitis Index (BI) and functional parameters were recorded. After treatment there was a significant reduction in the BL levels of IL-8 (1603.4 ± 331.2 pg/ml vs 1119.2 ± 265.3, $p = 0.01$) and MPO (1614.5 ± 682.3 µg/L vs 511.2 ± 144.2, $p = 0.05$), in cell numbers (250.6 ± 27.7 cells x 10^3/ml vs 186.3 ± 11.5, $p = 0.04$), neutrophil proportion (59.7 ± 14.3% vs 31.5 ± 10.1%, $p = 0.01$), SS (4.5 ± 0.6 vs 1.4 ± 0.5, $p = 0.01$), and BI (8.5 ± 0.8 vs 5.5 ± 0.7, $p = 0.007$). No significant changes were observed in the functional parameters. Treatment of stable COPD with high-dose inhaled steroids may induce changes in the levels of mediators and in the number and proportions of cells involved in AI.

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P2042
REDUCED LEVEL OF ENDOTHELIN-1 IN SMOKERS WITH AND WITHOUT CHRONIC BRONCHITIS

S. Torvaldsson[1], M. Laan[1], L Quarfordt[2], G. Riise[1], A. Lindén[1]. *[1]Lung Pharmacology Group, Dept of Respiratory Medicine and Allergology, Göteborg University, Gothenburg; [2]Dept of Infectious Diseases, Sahlgrenska University Hospital, Gothenburg, Sweden*

Airway mucociliary clearance is impaired by tobacco smoking and studies on canine tracheal epithelial cells have shown that endothelin-1 (ET-1) increases chloride secretion and enhances ciliary beat frequency *in vitro*. In this study, we examined whether tobacco smoking is associated with a reduced ET-1 level in the airways.

ET-1 protein levels were measured in bronchoalveolar lavage fluid (BALF) from 37 tobacco smokers with chronic bronchitis (CB), 10 asymptomatic smokers and 10 healthy never smokers using ELISA. The level of ET-1 was significantly reduced in BALF from asymptomatic smokers (1.5 ± 0.2 pg/ml, $p = 0.0002$) and smokers with CB (1.5 ± 0.1 pg/ml, $p < 0.0001$), compared with healthy never smokers (3.2 ± 0.5 pg/ml). No difference was noted between asymptomatic smokers and smokers with CB. In conclusion, the airways of tobacco smokers display a reduced level of ET-1 protein and this reduction is not related to CB. Additional studies will be required to determine whether a reduced ET-1 level leads to an impaired mucociliary clearance *in vivo*, as observed in the airways of tobacco smokers.

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P2043
STUDIES ON BRONCHIAL AND BRONCHOALVEOLAR LAVAGES IN SEVERAL BRONCHOPULMONARY PATHOLOGICAL PICTURES

C. Robalo Cordeiro, L. Mesquita, A.C. Pereira, L.C. Oliveira. *Center of Pneumology, Coimbra University – 3000 Coimbra, Portugal*

In order to assess different inflammatory cellular patterns and some signs of its bronchial and bronchoalveolar activations in several bronchopulmonary diseases, the authors studied the following populations: Bronchial asthma (BA) – 25 patients, mean age 38.5 ± 14.3 years, 2 smokers; Workers with Inhalatory Exposure to Isocyantes (EI) – 8 workers, 36 ± 9.4 years, 5 smokers; Healthy Heavy smokers (HS) – 19 individuals, 38.9 ± 6.4 years; Pulmonary Fibrosis (PF) – 8 patients, 47.5 ± 12 years, 2 ex-smokers; Healthy non-smokers (NS) – 4 individuals, 32.1 ± 11.1 years. The studies were made in Bronchial Lavage (BL) – 1st, 50 cc aliq., and in Bronchoalveolar Lavage (BAL) – 2nd and 3rd, 50 cc aliq., and included Neutr., Eos. and Mast cells (% and per ml), and dosages of Mieloperoxidase (MPO) – µg/l, Eosinophilic cationic protein (ECP) – µg/l and Triptase (TPT) – µg/ml. From the results the authors point out: a) positive linear regression, in BL, between Eos% and ECP ($p = 0.0001$ and $r = 0.67$), and between Neutr % and MPO ($p = 0.0316$ and $r = 0.36$) in all groups; b) higher Eos% and ECP in BL of BA regarding all other groups ($p = 0.0155$ and $p < 0.05$); c) higher number of Neutr/ml and Eos/ml in BAL of PF patients regarding all other groups; d) ECP in BAL only dosable in EI workers. As conclusions, we may suggest the interest 1 – of differentiated studies (BL *vs* BAL) assessing predominantly bronchial pathologic pictures (BA) and more distal diseases (PF and EI), and 2 – of studying, in lavage sampling, signs of cellular activation.

P2044
EFFECT OF A CIGARETTE WHICH DOES NOT BURN TOBACCO ON PHENOTYPICAL MARKERS OF ALVEOLAR MACROPHAGES FROM HEAVY SMOKERS

Takeshi Umino[1], Lidia S. Mansoulova[1], C. Magnus Skold[2], Samvel J. Pirruccello[1], John R. Spurzem[1], Stephen I. Rennard[1]. *[1]Depts of Internal Med and Path/Micro, University of Nebraska Medical Center, Omaha NE, United States; [2]Dept of Thoracic Med, Karolinska Hospital, Stockholm, Sweden*

Combustion of a cigarette causes release of numerous toxic substances. The prototype smokeless cigarette (Eclipse®) is designed to heat tobacco rather than burn it in order to extract nicotine and flavor. To determine if this device results in reduced airway inflammation, we performed bronchoalveolar lavage (BAL) in heavy smokers (>40 cigarettes/day) before and after two months of use of this product. Eighteen smokers were entered and twelve completed. Eight non-smokers were evaluated as controls. Phenotypic markers on alveolar macrophages (AM) were evaluated by a flow cytometrical method (Umino, T. *et al.* Eur Respir J 1999; in press). CD11c expression on AM was higher in smokers compared to non-smokers (8.2 ± 1.0 vs 1.5 ± 0.4, $P < 0.01$), and it decreased significantly after two months of use of the smokeless cigarette (5.9 ± 0.6, $P < 0.01$). CD71 expression on AM was lower in smokers than non-smokers (7.8 ± 1.3 vs 22.0 ± 4.0, $P < 0.05$), and there was a trend toward increase after the use of smokeless cigarette, although it was not significant (8.5 ± 0.9, $P = 0.57$). This smokeless cigarette partially normalized the alterations in AM markers caused by conventional cigarette smoking, suggesting it may be a reduced stimulus for the modification of AM function.

P2045
ASSESSEMENT OF BRONCHOALVEOLAR LAVAGE FLUID IN CHRONIC BRONCHITIS PATIENTS

E. Danila[1], L. Jurgauskiene[2], R. Malickaitė[2], M. Aviženienė[2], B. Šatkauskas[1]. *[1]Clinic of Pulmonology and Allergology, [2]Department of Immunology of Heart Surgery Clinic, Vilnius University, Vilnius, Lithuania*

Cell differentials and lymphocyte surface markers in BLF and ALF from 17 healthy nonsmokers (HN), 14 healthy smokers (HS), 7 nonsmokers chronic bronchitis patients without (CBN) and 10 patients with exacerbation (CBNe), 11 smokers chronic bronchitis patients without (CBS) and 9 patients with exacerbation (CBSe have been investigated.

	AM	L	N	E	CD4	CD8	CD4/CD8
B HN	83 ± 6	12 ± 5	5 ± 2	0.3 ± 0.3	42 ± 11	25 ± 10*	1.9 ± 0.5*
HS	80 ± 5	13 ± 4	6 ± 3	1 ± 1	39 ± 12	41 ± 11	1.0 ± 0.1
CBN	76 ± 8	18 ± 8	6 ± 1°	0.5 ± 0.6	47 ± 12	34 ± 20	1.8 ± .
CBS	78 ± 6	14 ± 7	7 ± 2°	1 ± 1	30 ± 11	39 ± 15	0.9 ± 0.2
CBNe	57 ± 24	17 ± 6	26 ± 27#	0.6 ± 0.3	33 ± 8	33 ± 5	1.0 ± 0
CBSe	66 ± 13	15 ± 6	19 ± 13**	0.7 ± 0.3	39 ± 13	41 ± 15	1.1 ± 0.5
A HN	82 ± 5	13 ± 7	5 ± 2	0.3 ± 0.5	47 ± 11*	32 ± 10*	1.6 ± 0.5
HS	80 ± 5	14 ± 3	6 ± 2	0.4 ± 0.6	36 ± 9	47 ± 15	0.9 ± 0.3
CBN	70 ± 10	21 ± 7	8 ± 3	1 ± 1	53 ± 21	34 ± 23	2.4 ± .
CBS	82 ± 4	12 ± 4°	6 ± 1°	0.7 ± 1	33 ± 8	26 ± 13	0.8 ± 0
CBNe	62 ± 13	21 ± 8#	16 ± 14#	1 ± 1	45 ± 15	38 ± 12	1.3 ± 0
CBSe	60 ± 14	17 ± 6	22 ± 17	1.0 ± 0.6	48 ± 14	45 ± 15	1.2 ± 0

B – bronchial, A – alveolar lavage fluid. *$p < 0.01$ between HN and HS. #$p < 0.05$ between HS and CBNe. °$p < 0.05$ between CBN and CBNe. **$p < 0.05$ between HS and CBSe. °$p < 0.05$ between CBS and CBSe.